Mass spectrometry of ribosomes and ribosomal subunits.

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Nanoflow electrospray ionization has been used to introduce intact Escherichia coli ribosomes into the ion source of a mass spectrometer. Mass spectra of remarkable quality result from a partial, but selective, dissociation of the particles within the mass spectrometer. Peaks in the spectra have been assigned to individual ribosomal proteins and to noncovalent complexes of up to five component proteins. The pattern of dissociation correlates strongly with predicted features of ribosomal protein-protein and protein-RNA interactions. The spectra allow the dynamics and state of folding of specific proteins to be investigated in the context of the intact ribosome. This study demonstrates a potentially general strategy to probe interactions within complex biological assemblies.

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Proteins that interact with GroEL and factors that affect their release

This table is simply designed to show the diverse nature of GroEL substrates. It is not intended to be an all inclusive bibliographic reference. Citations given are generally for the earliest documentation for that substrate.

If you know of substrates not listed below, please forward that information to Jeff Seale

The first 31 entries in this database were compiled by Boris Gorovits

N/A in the Release requirements means that the cited reference may not have determined this information

Protein	Release requirements	Reference
alcohol oxidase	MgATP	(1)
alpha-glucosidase	MgATP	(2)
alpha-lactalbumin	N/A	(3)
aspartate aminotransferase	MgATP + GroES or MgATP	(4)
barnase	MgATP or none	(5)
beta-lactamase precursor	MgATP + GroES	(6)
chloramphenicol acetyltransferase	GroE system not required	(7)
chloroplast precursor protein	GroES + MgATP; casein + MgATP	(8)
citrate synthase	GroES + MgATP	(9)
CRAG	ATP	(10)

cyclophilin	GroES + MgADP	(11)
Cu,Zn superoxide dismutase	N/A	(12)
dihydrofolate reductase	MgATP required; GroES helps	(13)
dodecameric glutamine synthase	MgATP; or MgADP + GroES	(14)
E2 inner core bovine mitochondrial branched chain a-keto acid dehydrogenase	GroES + MgATP	(15)
F(ab) fragments	GroES + MgATP	(16)
glucose-6-phosphate dehydrogenase	none, or MgATP	(17)
granulocyte RNase	MgATP	(18)
lactate dehydrogenase	MgATP or MgAMP-PNP	(19)
luciferase	this study in vivo	(20)
malate dehydrogenase	MgATP; K+ is not obligatory	(21)
non-glycosylated invertase	MgATP; GroES helps; glyco form does not interact	(22)
ornithine transcarbamylase	GroES + MgATP ATP analogues with GroES do not work	(23)
phytochrome photoreceptor	MgATP	(24)
RNA polymerase	GroES + MgATP (?)	(25)
RUBISCO	GroES + MgATP	(26)
RNA polymerase, sigma subunit	N/A	(27)
ssDNA binding protein	N/A	(28)
tryptophanase	ATP, ADP, AMP-PNP	(29)
tubulins	GroES + MgATP	(30)
yeast enolase	MgATP; or MgADP + GroES	(31)

Taka-amylase A	GroES + ATP or ADP	(32)
E. coli B-galactosidase	GroEL reduces aggregation, GroEL+ ATP or AMP-PNP leads to aggregation	(33)
rhodanese	GroES + MgATP & K+	(34)
carbonic anhydrase II		(35)
prion protein PrPc	not determined	(36)
NiFe hydrogenase 3 precursor	not determined	(37)
glycerol dehydrogenase	ATP increases kinetics; GroES not required	(38)
trichosanthin	Mg, ATP	(39)
staphylococcal nuclease	ATP accelerates refolding; ATP+GroES maximal refolding	(40)

This information is accurate to the best of my knowledge. However, you should check the references cited for a more complete understanding of these substrates and their interactions with GroEL.

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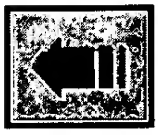

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1: *Proc Natl Acad Sci U S A* 1995 Jul 3;92(14):6459-63

Characterization of the VHL tumor suppressor gene product: localization, complex formation, and the effect of natural inactivating mutations.

Duan DR, Humphrey JS, Chen DY, Weng Y, Sukegawa J, Lee S, Gnarra JR, Linehan WM, Klausner RD

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The human VHL tumor suppressor gene has been implicated in the inherited disorder von Hippel-Lindau disease and in sporadic renal carcinoma. The homologous rat gene encodes a 185-amino acid protein that is 88% sequence identical to the aligned 213-amino acid human VHL gene product. When expressed in COS-7 cells, both the human and the rat VHL proteins showed predominant nuclear, nuclear and cytosolic, or predominant cytosolic VHL staining by immunofluorescence. A complicated pattern of cellular proteins was seen that could be specifically coimmunoprecipitated with the introduced VHL protein. A complex containing VHL and proteins of apparent molecular masses 16 and 9 kDa was the most consistently observed. Certain naturally occurring VHL missense mutations demonstrated either complete or partial loss of the p16-p9 complex. Thus, the VHL tumor suppressor gene product is a nuclear protein, perhaps capable of specifically translocating between the nucleus and the cytosol. It is likely that VHL executes its functions via formation of specific multiprotein complexes. Identification of these VHL-associated proteins will likely clarify the physiology of this tumor suppressor gene. Copyright 1999 Academic Press.

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1: *Science* 1995 Sep 8;269(5229):1402-6

Inhibition of transcription elongation by the VHL tumor suppressor protein.

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Germline mutations in the von Hippel-Lindau tumor suppressor gene (VHL) predispose individuals to a variety of tumors, including renal carcinoma, hemangioblastoma of the central nervous system, and pheochromocytoma. Here, a cellular transcription factor, Elongin (SIII), is identified as a functional target of the VHL protein. Elongin (SIII) is a heterotrimer consisting of a transcriptionally active subunit (A) and two regulatory subunits (B and C) that activate transcription elongation by RNA polymerase II. The VHL protein was shown to bind tightly and specifically to the Elongin B and C subunits and to inhibit Elongin (SIII) transcriptional activity in vitro. These findings reveal a potentially important transcriptional regulatory network in which the VHL protein may play a key role.

Comment in:

- Science. 1995 Sep 8;269(5229):1400-1

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